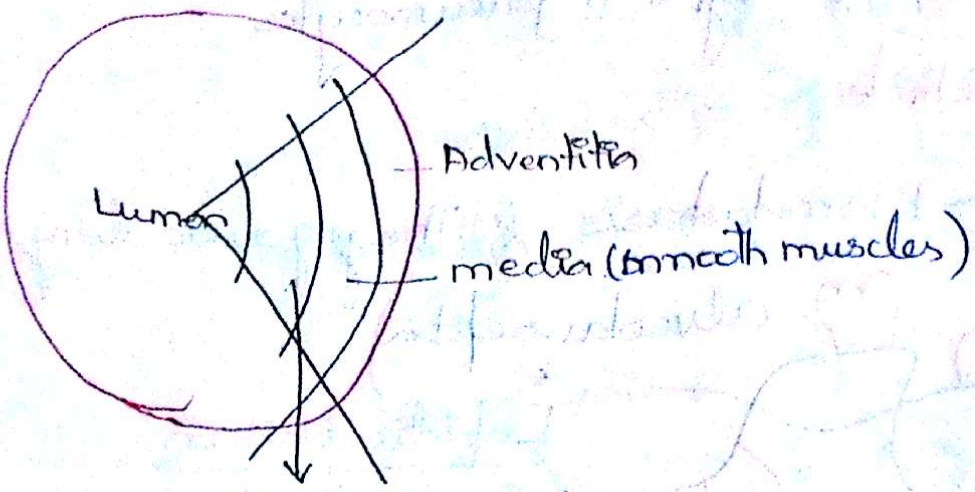
CVS



Intima
One/2 layers of endothelial cells

Neo-intima endothelial cells + New smooth muscle cells
during injury, inflammation, grafting.

Atherosclerosis - Intimal based plaques.

Risk factors

Non-modifiable
→ Old age
→ male
→ stress (Type-A)
Hyperhomocystinuria

Modifiable
- Obesity
- DM
- HTN

Infections-
CMV + Herpes + chlamydia.

Widely accepted Hypothesis - Endothelial injury

Intimal

Cytokines

Recruit Smooth muscles | Inflammation (macrophages)

Oxidized LDL + macrophages / Smooth muscles → Foam cells

↓

Fibrous plaque

↓

Fatty streak (earliest feature visible)

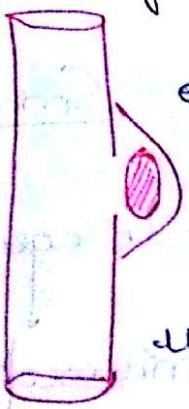
m/c site - Abdominal aorta

Least c/c site - Circle of willis

Aneurysm - Dilatation of Blood wall / Heart wall

a) True aneurysm: All 3 layers of Blood vessel wall

b) Pseudo Aneurysm - only one wall (intimal) Tear



extravascular Hematoma

m/c Cause - Post myocardial cardiac wall ruptures

c) Mycotic aneurysm - m/c a/w Staphylococcus

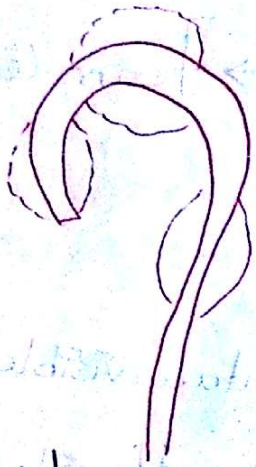
m/c site - Femoral Artery

4) Luetic aneurysm - Syphilis (Tertiary stage)

Intimal wrinkling - Tree Bark appearance

m/c site - Ascending aorta (Root).

AR → enlarged Heart chamber Cor bovinum



Ascending A
Hypertension

Arch

Descending

Atherosclerosis.

Aortic dissection - 40-60 years.

Dissection into T. media.

m/c Cause aortic dissection → HTN

m/c Histology - cystic medial degeneration:
[marfans, OI, EDS]

Vasculitis

Large	Medial	Small
1) Giant cell arteritis	1) PAN	Wegener's
2) Takayasu.	No ANCA	microscopic polyangitis
	all stages of inflammation in all vessels	Churg-strauss

Large | Medium | Small

Giant cell arteritis | Kawasaki | Churg strauss

Segmental Biopsy | anti endothelial Ab | eosinophilic granuloma

granulomatous fragmentation of internal elastic lamina | | P-ANCA.

M/c Cause of vasculitis:

a) Pediatric – Henoch Schonlein purpura.

b) Adult – Giant cell arteritis.

c) m/c COD in ped – Kawasaki.

II Takayasu arteritis - m/c site - Subclavian arteritis.

m/c Cause of mononeuritis multiplex
Diabetes mellitus (Ind/world)
→ amongst Vasculitis - PAN.
Hairy cell leukemia – PAN.
Anti endothelial antibodies - Kawasaki.

Coronary Artery disease

Significant Obstruction >75%

Risk factors -

1) hs-CRP.
2) Total cholesterol
   HDL
3) ApoB / ApoA.
4) LDL
5) HDL.

m/c site

Left anterior descending artery.
↓
mI (LV)

Gross - upto 12 Hrs no
finding / occasional microscopy

TTC Assay - Triphenyl Tetrazolium chloride assay.

TTC.

Normal → LDH. + TTC. ↓ Reduced Colour ↓ normal.

M.I → all LDH Released into Serum. ↓ (no LDH to combine) Pale colour ↓ M.I.

Biochemical markers -

earliest — myoglobin.

m. sensitive/specific - Troponin I > T.

↑ 2-4hrs → 7-10days.

Re-infarction - CK-MB.

Flipping of LDH - Normal LDH 2 > 1

Flip pattern - LDH 1 > 2.

Gross

12hrs Occasional dark mottling

12-24Hrs - established dark mottling

1-3days - yellow-tan-infarct.

3-7days - Hyperaemic Borders

2months - Scarring complete.

Histopathology

4Hrs - waviness of fibres.

12-24hrs - Coagulative necrosis. early neutrophilic infiltrate

[1-3days max Neutrophilic infilt.]

7-10days - early granulation tissue

10-14days - max. granulation tissue.

EM

1st ½hour - Mitochondrial Swelling.

½hour - Large flocculent amorphous densities.

Rheumatic Heart Disease.

GABH: 1, 3, 5, 6, 18

Pharyngitis $\xrightarrow{3\%}$ RHD

Valve m/c - mitral

4/c - pulmonary

Aschoff Bodies:

Swollen Collagen
Plasma cells
Histiocytes - Caterpillar like nuclear chromatin } Diagnostic of RHD
↓
Anitschow cells

→ No epithelioid cells
No langhans giant cells

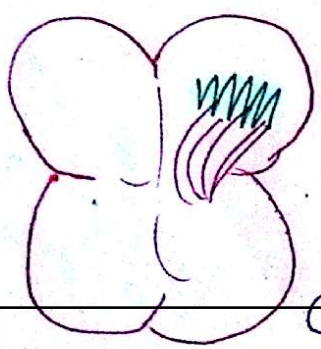
Aschoff Bodies - myocardium (m/c) - Perivascular areas.
Pericardium
endocardium

Acute RHD:

m/c - mitral Regurgitation.

Chronic mitral stenosis

overall - mitral stenosis.



① Atrium posterior wall
Subendocardial patches
Aschoff Bodies } Mac Callum plaques

Commonly seen in Chronic RHD

[illegible] cause of Death – endocarditis

Chronic RHD – myocarditis.

Overall – myocarditis

RHD	NBTE ↑(Coagulable) (malignancy, ↑estrogen)	LSE	I.E)
→ Small	Small (NBTE > RHD) more prone for embolisation.	medium verrucous. flat	Large
sterile	sterile.	Sterile	Non-sterile.
along line of closure of wall	along line of closure of wall	either side of wall. Lower Surface > up. → pocket of walls. → Destructive.	Both surfaces upper > lower. max embolization, most destructive

One liners –

→ Dilated Cardiomyopathy – a/w Largest Titin (30,000).

→ Ⓡ wall.

→ Arrhythmogenic Ⓡ ventricular Cardiomyopathy.

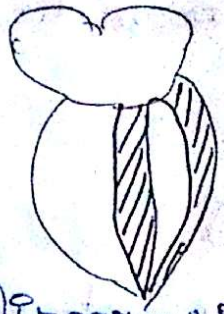
a/w plakoglobin protein mutation.

→ LV Hypertrophy – ↑ osteoglycin (OGN).

HOCM :- m/c cause of Sudden death in athletes HOCM

m/c mutation — β mHC gene (myosin heavy chain).

Types:



- Banana like cavity.

m/E: Disarray of myofibres.
Heller - Skelted disarray

Myocarditis - Viral infections.

m/c - Parvovirus - B19 + Herpes > Coxsackie.

Parasite - m/c - Trichinella Spiralis.

m/c Cause of Hemorrhagic pericarditis → malignancy
m/c Cause - Lung Ca.

m/c Splen tumour → Benign Cavernous Hemangioma
Rest all → malignancies.

m/c Tumour of Heart - metastasis
m/c - 1° lung.
m/c site - pericardium.
Cardiac tumours

Pediatric

m/c i malignancy
Rhabdomyosarcoma.
1° Benign Rhabdomyoma.

Adults

m/c i malignancy - Angiosarcoma.
m/c Benign i - Cardiac myxoma.